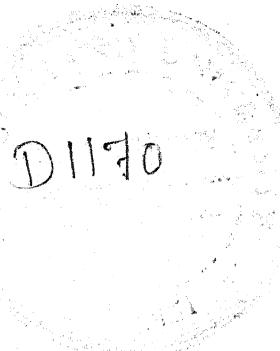


**"SCREENING AND PREVALENCE OF
HYPERTENSION IN SCHOOL AGE CHILDREN
AND YOUNG ADULTS AND TREATMENT
IN BUNDELKHAND AREA"**

**THESIS
FOR
DOCTOR OF MEDICINE
(INTERNAL MEDICINE)**



**BUNDELKHAND UNIVERSITY
JHANSI(U.P.)**

2005

DHARMENDRA KUMAR

DEDICATED
TO
MY PARENTS

CERTIFICATE

This to certify that the work entitled "SCREENING AND PREVALENCE OF HYPERTENSION IN SCHOOL AGE CHILDREN AND YOUNG ADULTS & TREATMENT IN BUNDELKHAND AREA", Which is being submitted as a thesis for M.D. (Medicine) Examination, 2005 of Bundelkhand University, has been conducted by Dr. Dharmendra Kumar in the department of Medicine, M.L.B. Medical College, Jhansi.

He has put the necessary stay in the department as per University regulations.

(Dr P.K. Jain)
M.D. MNAMS

Prof. and Head,
Department of Medicine,
M.L.B. Medical College,
Jhansi

Dated:

30/10/04

CERTIFICATE

This to certify that the work entitled "SCREENING AND PREVALENCE OF HYPERTENSION IN SCHOOL AGE CHILDREN AND YOUNG ADULTS & TREATMENT IN BUNDELKHAND AREA", Which is being submitted as a thesis for M.D. (Medicine) Examination, 2005 of Bundelkhand University, has been carried out by Dr. Dharmendra Kumar under my direct supervision and guidance. The techniques embodied in this thesis have been undertaken by the candidate himself and the observations recorded have periodically been checked and verified by me.


(Dr. R.C. Arora)

M.D. D.Sc

Former Prof. and Head,
Department of Medicine,
M.L.B. Medical College,
Jhansi

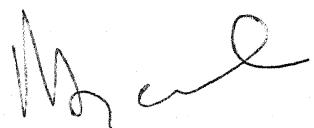
(GUIDE)

Dated:

30/10/04

CERTIFICATE

This to certify that the work entitled "SCREENING AND PREVALENCE OF HYPERTENSION IN SCHOOL AGE CHILDREN AND YOUNG ADULTS & TREATMENT IN BUNDELKHAND AREA", Which is being submitted as a thesis for M.D. (Medicine) Examination, 2005 of Bundelkhand University, has been carried out by Dr. Dharmendra Kumar under my direct supervision and guidance. The techniques embodied in this thesis have been undertaken by the candidate himself and the observations recorded were checked and verified by me from time to time.



(Dr. Navneet. Agarwal)
M.D.

Asst. Professor,
Department of Medicine,
M.L.B. Medical College,
Jhansi

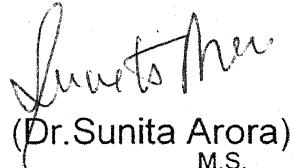
(Co-GUIDE)

Dated:

30/10/01

CERTIFICATE

This to certify that the work entitled "SCREENING AND PREVALENCE OF HYPERTENSION IN SCHOOL AGE CHILDREN AND YOUNG ADULTS & TREATMENT IN BUNDELKHAND AREA", Which is being submitted as a thesis for M.D. (Medicine) Examination, 2005 of Bundelkhand University, has been carried out by Dr. Dharmendra Kumar under my direct supervision and guidance. The techniques embodied in this thesis have been undertaken by the candidate himself and the observations recorded were checked and verified by me from time to time.



(Dr. Sunita Arora)
M.S.

Dated:

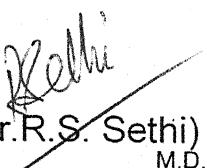
30/10/04

Associate Professor,
Department of Obst. &
Gynaecology,
M.L.B. Medical College,
Jhansi

(Co-GUIDE)

CERTIFICATE

This to certify that the work entitled "SCREENING AND PREVALENCE OF HYPERTENSION IN SCHOOL AGE CHILDREN AND YOUNG ADULTS & TREATMENT IN BUNDELKHAND AREA" , Which is being submitted as a thesis for M.D. (Medicine) Examination, 2005 of Bundelkhand University, has been carried out by Dr. Dharmendra Kumar under my direct supervision and guidance. The techniques embodied in this thesis have been undertaken by the candidate himself and the observations recorded were checked and verified by me from time to time.


(Dr. R.S. Sethi)
M.D.

Dated:

30/10/04

Asst. Professor,
Department of Paediatrics,
M.L.B. Medical College,
Jhansi

(Co-GUIDE)

ACKNOWLEDGEMENT

It would take pages, to acknowledgement everyone who in some time who in some way or the other helped me along my path reaching this goal, but certain individuals need special thanks for valuable help.

First and foremost, with all my heart I would like to express my deepest gratitude and privilege for having been associated with **Dr. R.C. Arora M.D. D.Sc.** Former Principal and Dean, Professor and Head, Department of Medicine, Maharani Laxmi Bai Medical College, Jhansi. For his enlightened guidance which stressed me towards the harbour of success. I am greatly thankful for the ceaseless attention, he has paid to my problems, always sparing a few movement for me even amidst the busiest of schedules without which this work would not have seen the light of day.

"The habits of punctuality, order, diligence, determination and concentration are the key of success". That is what has been inculcated into us by our honorable Asst. Professor and my co-guide, **Dr. Navneet Agarwal M.D.**, Department of Medicine , M.L.B. Medical College , Jhansi .

I am very thankful to **Dr. Sunita Arora M.S.** Asst. professor Department of Gynae. & Obstetrics , whose time to time highly valuable inputs help me a lot .

I am greatly thankful to **Dr. P.K. Jain M.D. MNAMS**, Prof. & Head of Department of Medicine M.L.B. Medical College, Jhansi. His sense of precision , inlinching , tenacity , passion for reason ,

compassion toward patients , knowledge and experience was a constant source of inspiration to me.

I am also grateful to **Dr. Praveen Kumar Jain M.D. DM.** Asst. Professor of Cardiology, who always helped me in every possible way to make this study a success.

I am also grateful to **Dr. R.S.Sethi M.D.** (Paediatrics) who always helped me in every difficult moment . He also solved my paediatrics problems .

I am also greatly indebted to **Dr. N.S. Sanger M.D. D.M.** Nephrology , Asst. Professor in Medicine and **Dr. Gynendra Kumar M.D.(Psychiatry)**, Asst. Professor Department of Medicine , for giving me inspiration and encouragement .

I am very thankful to my wife **Dr. Archana Singh** who by her boundless cooperation, unconditional and firm support has been a constant source of inspiration.

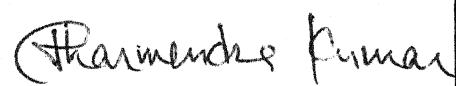
I am indebted to my parents for their inspiration, moral support, love and encouragement throughout my studies.

I am thankful to my brother and Bhabhi for their attention and help us when needed.

I would like to express my thanks to **Mr. Shashank Jain** Chief of Net Sol computers for the pain, he took in helping me prepare a presentable and near work to submit.

Dated :

30/10/04



(Dr. Dharmendra Kumar)

CONTENTS

S.No.	CHAPTER	Page no.
1.	INTRODUCTION	1-10
2.	REVIEW OF LITERATURE	11-27
3.	AIMS AND OBJECTIVE	28
4.	MATERIAL AND METHODS	29-39
5.	OBSERVATIONS	40-50
6.	DISCUSSION	51-56
7.	CONCLUSION	57-58
8.	BIBLIOGRAPHY	59-63

INTRODUCTION

INTRODUCTION

Conceptual Definition: Blood pressure is a physiological variable and is subject to be influenced by many factors viz environment, emotional state and drugs etc. An elevated arterial pressure is probably the most important health problem. It is common, asymptomatic, readily detectable usually easily treatable and often leads to complications if untreated. Epidemiological studies indicate a strong correlation between elevated arterial pressure and cardiovascular morbidity and mortality. Since the publication of the 1993 issue of pediatric clinics of North American that was devoted entirely to childhood, hypertension much has been published relating to development, investigation and treatment of elevated blood pressure in children and adolescents. The incorporation of blood pressure measurement into routine pediatric examination has enabled both the discovery of significant asymptomatic hypertension secondary to a previously undetected disorder and conservation that mild

elevation in blood pressure during childhood are more common than previously recognized, particularly in adolescents.

Kaplan in 1983 proposed that conceptual definition of hypertension be "that level of blood pressure at which the benefits (minus the risk and costs) of action exceeded the risk and costs (minus the benefits) of inaction".

Blood pressure is considerably lower in children than adults but almost always increases steadily throughout the first two-decade of life. During preschool years blood pressure begins to follow a pattern. Children at a given percentile of blood pressure distribution tend to maintain that approximate value relative to their peer group as they grow older; with correlation ranging from 0.30 to 0.66 for systolic blood pressure and 0.12 to 0.57 for diastolic in childhood and adolescence. The pattern continues from adolescence into adult life which support the hypothesis that essential hypertension begin in childhood however it is not currently possible to identify which children will have essential hypertension as adults.

A number of factors known to be associated with hypertension in adults have also been associated with higher level of blood pressure in children and adolescent. A direct relation between weight and blood pressure has been documented as early as five years of age and is more prominent in the second decade. Height is independently related to blood pressure at all ages. Sex and race do not have the same impact on blood pressure in children as adults, no significant differences in blood pressure have been found in comparisons of whites, Blacks, Hispanics and South East Asian until adolescence. Even the differences are small and vary among epidemiological studies, blood pressure is slightly higher in boys then in girls during the first decade of life, this difference begins to widen around the onset of puberty, and blood pressure is significantly higher in young men by the end of teenage years. These differences do not appear to be related to development changes in concentrations of follicle-stimulating or utilizing hormone.

A familial incidence on blood pressure can be identified early in life, children from families with hypertension tends to have higher blood pressure than children from normotensive families.

There are significant correlation in blood pressure and cardiovascular risk factors between parents and their children. Sibling of children with high blood pressure have significantly higher blood pressure than siblings of children with low blood pressure.

In children normal blood pressure is defined as systolic and diastolic blood pressure less than 90th percentile for age and sex. High normal blood pressure defined as an average systolic and diastolic blood pressure of greater than or equal to 90th percentile but less than the 95th percentile. And hypertension is defined as average systolic or diastolic blood pressure of greater than or equal to 95th percentile for that age and sex and measured at least three separate occasions.

According to JNC VII: In case of young adults systolic blood pressure <120mmHg and diastolic blood pressure <80mmHg is considered normal. In prehypertensive the range of

systolic blood pressure 120-139 mmHg and diastolic blood pressure 80-89 mmHg.

Grade

Stage I hypertension: - Systolic BP 140-159 mmHg and diastolic BP 90-99 mmHg.

Stage II hypertension: - Systolic BP ≥ 160 mmHg and diastolic BP ≥ 100 mmHg.

In pediatric age group we categories hypertension in two category:

1. Significant hypertension- In 6-9 years of age group systolic BP > 121 mmHg and diastolic BP > 77 mmHg.

In 10-12 years age group systolic BP > 125 mmHg and diastolic BP > 81 mmHg.

2. Severe hypertension

In age group 6-9 years systolic BP > 129 mmHg and diastolic BP > 85 mmHg.

In age group 10-12 years systolic BP > 133 mmHg and diastolic BP > 89 mmHg.

In essential hypertension in about 95% of cases no cause can be established. In cases of secondary hypertension 5% of patients with hypertension have specific cause.

Ingelfinger: 1994 evaluated the young patients and found that approximately 60 to 80% of secondary hypertension in children was caused by renal parenchymal disease and approximately 5 to 25% of children with secondary hypertension are said to have renovascular disease. Estimate from several BP surveys over past decade, suggest that up to 15% of the adult population have definite or established hypertension and that an almost equal % have labile hypertension.

Thirty year's surveillance in the **Framingham** study did not find any consistent trend in the incidence of new onset hypertension in United States, although two third of the study cohort eventually developed hypertension. The overall incidence of hypertension in urban population of Delhi was reported as 1.22%, while the incidence in rural around Delhi was reported to be 0.28% (Males 0.38% and Females 0.24%). The cut- off point

used for hypertension in the rural population in the above study was SBP \geq 160mmHg and DBP \geq 90mmHg.

Comparison of the incidence of hypertension, systolic BP \geq 160 mmHg and diastolic BP \geq 95 mmHg. In white men and women shows an approximately 5% increase for each 10 years interval of age at base line, except in the 65 to 74 years old age group. The incidence among black was at least twice that among **whites**. The higher incidence is seen in the 55 to 64 years old and 65 to 74 years old age groups.

Prevalence: - The prevalence of hypertension depends on both the racial composition of population studied and the criteria used to define the condition. In white suburban population like in **Fragmingham** study, nearly one fifth have BP more than 160/95 mmHg.

There is no available data to define the frequency of **secondary hypertension** in the general population, although in middle aged males it **has been reported to be 6%**.

National Health and nutrition examination survey (NHANES) in the United States have reported a prevalence rate

20% of the entire US population in 1991. The prevalence rate varies from 4% in the age group 18 to 24 years to 60% in the age group 65 to 74 years.

In a cohort of Junior- High- School students the prevalence of hypertension **fell from 4.2% at an initial examination to 1.1% of after only one repeated examination.**

There are no data on the prevalence of essential hypertension in children.

Review of the cases of hypertension before adulthood have tended to rely on data from medical center referral patients with severe elevation of blood pressure, and these patient have secondary cause of their hypertension. Never the less, it has become clear since the incorporation of blood pressure measurement into the routine physical examination that in most cases mild to moderate hypertension in students in Junior and Senior high school is not associated with secondary disease and that essential hypertension should be an important consideration. Renal disease continues to be the most common cause of hypertension when a diagnosis is made in this group, but

secondary cause are found much less frequency than in younger patients.

Sinaiko found the prevalence of significant hypertension (either essential or secondary) to be 2% among 14,000 school age children age 10-15 up to 80% of hypertensive adolescent have essential hypertension. However a secondary cause found in 80% of children younger than 10 year old. At least 90% of hypertension adults have no underlying secondary cause.

Denials found a 14% prevalence of LVF in adolescent who had essential hypertension. Secondary hypertension include renovascular disorder (12%), endocrine disorder 8% and variety of rare condition 2% such as **coarctation** of the aorta.

Classification of School age Children

Category	Significant hypertension		Severe hypertension	
	Systolic BP (mmHg)	Diastolic BP (mmHg)	Systolic BP (mmHg)	Diastolic BP (mmHg)
6-9 years	>121	>77	>129	>85
10-12 years	>125	>88	>133	>89

Classification in young adults. according to JNC VII**young adults:-**

Category	Systolic BP(mmHg)	Diastolic BP(mmHg)
Normal	<120	<80
Pre-hypertensive	120-139	80-89
Hypertension		
Stage I	140-159	90-99
Stage II	≥ 160	≥ 100

REVIEW
OF
LITERATURE

REVIEW OF LITERATURE

An overview of the natural history of hypertension indicates a combination of hereditary and environmental factors set into motion transient but repetitive perturbations of cardiovascular homeostasis. This is the stage of prehypertension. These elevations of pressure are not severe enough to be defined as abnormal but high enough to begin a cascade that, over many years, leads to pressures in the abnormal range. This would be the stage of early hypertension. At this stage some people might, by modification of life-style and diets, be able to revert the process and go back normotension. The majority, however goes on to have established hypertension. This eventually leads to target organ damage and disease. It is now well established that higher the blood pressure, the greater the morbidity and mortality. Though occasionally some people with high blood pressure never have trouble, there is no way to identify those who will have an uncomplicated course. Similarly there is no way to identify those hypertensive who will have a rapidly accelerating phase or malignant hypertension. But the majority will slowly but progressively develop cardiovascular and other complications.

The role of hypertension is probably underestimated from morbidity and mortality statistics which are largely based on death certificates, when a patient dies from a stroke, heart attack or renal

failure all these clinical conditions have a direct correlation with uncontrolled hypertension.

In the natural course of hypertension there are certain clues that may predict that the patient is in the pre-hypertensive phase. These would include exaggerated rises in BP, during stress (**Light et al 1992**), or exercise(**Molineux and Steptoe 1988**). In a group 341 people who had normal (< 140/90 mmHg) resting BP but a rise during treated exercise test to above 225/90 mmHg . The relative risk of developing a high resting BP. Over the next 32 months was 228 times higher then among those with a lesser rise during the exercise test (**Willson and Meyer 1981**).

Another indicator of the patients being in the pre hypertensive phase are pressures that are in the higher range of normal. As reported by **Kotchen et al (1992)** , on the **Framingham study Cohrt**; the BP tends to track over many years, remaining in the same relative position over times. After an initial regression towards the mean between the first examination and the second, 2 years later, those in each segment of BP. Tend to remain in that segment this is followed by gradual rise over the next 14 years.

Per- hypertension is also characterised by presence of number of causal or coincidental features. In the 30 years observation of the off

springs of the **Framingham Cohort** , **Garrison et al (1987)** found the major contributors of hypertension bed sides age were adiposity, heart rate, alcohol intake and triglyceride levels. Phosphorus has negative correlation with the incidence of hypertension. **Garrison et al (1987)** also found that for development of hypertension the stronger predictor was the previous level of BP.

The Australian therapeutic trial **Mng Committee (1982)** concluded that treatment of hypertension should only begin often it has been confirmed by a number of reading, only then therapy is began. If the second set of readings is considerably lower and the patients is free of obvious vascular complication the patient should be advised to adhere to a healthy life style and to return few months for repeat measurement. 12.8% of patients whose diastolic BP, averaged above 95 mmHg on two initial readings had a subsequent fall in DBP below 95. An even larger portion (47.5) of those who entered the trial with DBP above 95, who received only placebo tables for the next 3 years maintained their average DBP below 95 mmHg. A significant portion remained below 90mmHg while on placebo including 11% of those whose intitial DBP was as high as 105 mmHg to 109 mmHg.

The terms of age of onset **Maxwell et al (1975)** noted the diagnosis of primary hypertension with great certainty in 1128 patients of

there the onset of an elevated BP was documented to be below age 30 in 12 % and above 50 in only 7%. The majority of primary hypertension cases therefore lie in the 30-50 years age group.

In the **Australian** the rapentic trial over 1600 adults with DBP of 95 to 105 mmHg initially free of known cardiovascular disease were kept on placebo for an average of 3 years over this relatively short period significantly increased morbidity and mortality occurred only in those whose DBP averaged 100 or higher during this interval. The average fall in BP was 14/11 mmHg and the DBP was below 95 in 47.5% of the patients at the end of the trail. Though the fall was greatest in those who lost weight there was a significant decrease in the average BP even among those who gained weight on placebo. The implications drawn from this trial were.

- Many patients not taking antihypertensive drug will have a significant fall in their BP often to levels considered safe and not requiring therapy.
- Patients, free of target organ damage and whose DBP are below 100 and certainly below 95 can safely be left off active drug therapy for at least a few years. If not treated, patients should be kept under close observation.

Helge Land et al 1980 conducted the Osiotrial . They included only uncomplicated patients free of target organ damage with a DBP ; below 110 mmHg and randomly divided them into non therapy and drug therapy groups about half of the non- treated group had a fall in DBP during the first 3 year. Few complications developed among those whose DBP was intially below 100 mmHg, where as 16.4% of those within initial DBP between 100 and 110 mmHg had cardiovascular complication.

Measurement of Blood Pressure: The variability of the pressure on repeated measurements is great considering the degree of variability found between single measurement made on different occasion, Pery and Miller (1992) concluded, perhaps, only one third to two thirds of people whose measured diastolic pressure exceeded 95 mmHg actually have pressures that high. In a general population single measurements of diastolic pressure exceeds 95 mmHg in approximately equal numbers of normotensives , borderline and hypertensive patients , more over one third of those who are usually in the hypertensive ranges are not identified.

In a study of **Schecter and Adler (1988)** on Bayesian analysis, the predictive value of two DBP reading above 90 for the presence of ' true DBP, above 90 is only 52 % . If the coverage of eight reading in above 90 the sensitivity or positive predictive value goes up but only to 73%. In another article **Conway (1986)** discussed the variability in BP in three different ways.

1. **Short term Variability.** This is affected by respiration and heart rate, which are under the influence of the autonomic nervous system.
2. **Day time variability:** This is mainly determined by the degree of mental and physical activity and is modified by baroreflexes that operate through adjustments in heart rate and peripheral resistance.
3. **Diurnal Variability:** This is substantial with an average fall in pressure of 20% during sleep and stimuli that decrease sympathetic nervous activity.

In a study by **Clark et al (1987)** the over riding influence of activity on diurnal variations was nicely demonstrated in a study of

461 untreated hypertensive patients whose BP was recorded with a portable non invasive device every 15 minutes during the day and every 30 minutes at night over a 24 hour interval. In addition five reading were taken in the clinic before and another five after the 24 hours recording. When the mean DBP reading for each of the 24 hours were plotted against each patients mean clinic BP, considerable variation were noted. The lowest pressures were recorded at night. The highest pressures were recorded near mid-day. The patients recorded in a diary where their BP were taken e.g. at home, work or other location and what they were doing at that time, with 15 choices of activity. When effects of the various combination of location and activity on the B.P. were analysed, variable effects relative to BP recorded while relaxing were seen when the estimated effects of various combinations of locations and activity were the subtracted from the individual reading obtained throughout the 24 hour period every little residual related to the time of day was found. The authors concluded that there is no important cicadian rhythms of blood pressure, which is independent of activity.

Racial Differences: Although blood pressure has been shown to be higher in black children compared with white children the

difference have not been believed to be clinically relevant and the current reference standards for children do not distinguish between racial or ethnic groups(**Berenson G, Wattigney W, Webberl Rep 111 : 3, 1996**). More recently in a study of 24,000 girls aged 9 to 10 years, **Daniels et al** reported that, although black girls had significantly higher systolic and diastolic blood pressure compared with white girls, stage of maturation accounted for the difference.

In a study of 2866 urban children aged 6 to 9 years, Asian boys had statistically significantly higher systolic and diastolic blood pressure compared with their hispanic and white but not black counterparts and Asian girls had significantly higher systolic and diastolic blood pressure compared with any other racial or ethnic group despite adjustment for age, weight and height (**Liuk, LevinsonS, public health rep. 11:65; 1996.**

Hypertension with obesity: The association of obesity and hypertension is believed to be a causal one, wherein obesity contributes not only to higher blood pressure in children and adolescents but also ahs been shown to have a significant predictive value for its association with other cardiovascular risk factors, such as cholesterol levels and serum lipoprotein ratios

between 13% and 36% of 12 to 17 year old adolescents are obese. Depending on gender and race, an additional 4% to 12% may be considered super obese. Likewise, a 54 % increase has occurred in the prevalence of obesity among children 6 to 11 year old. These data represent a 39% increase in prevalence of obesity compared with data collected between 1996 and 1970 (William D, Goings, Lohmant T et al 1992).

Essential hypertension: approximately 25% of the adult population in the united states has hypertension, with 90% being classified as having essential hypertension although the prevalence of essential hypertension is lower in children and adolescents than in adults, ample evidence supports the concept that the roots of essential hypertension extend back to childhood (Sinaiko A , 1989).

A familial and longitudinal studies of blood pressure show that a link exists between genetic and environment influences on blood pressure during childhood and the development of essential hypertension. This influence can be tracked to gestation and has been reviewed in depth by Iow and Barker 1995).

A familial influence on blood pressure can be identified early in life. Children from families with hypertension tends to have

higher blood pressure than do children from normotensive families (**Monger R , Gomes Mantion 1998**). A heterogenous group of factors contributes to risk for essential hypertension aside from genetic predisposition . These include reactivity of vascular smooth muscles and the kidney and interaction of reninangiotensin system, cardiac index, obesity and hormonal and environmental factors (**Jiang X, Srinivas , Bao w et al 1993**). One of the most promising recent developments has been the discovery of candidates genes for hypertension that highlight the role of the renin – angiotensin system (**Caulfield M, Lavendul , Farrall M, et al 1994**).

Cardiovascular Effects of Hypertension:- In adults , elevated blood pressure accelerates the development of coronary artery disease (CAD) and contributes significantly to the pathogenesis of cerebrovascular accidents , hearts failure and renal failure. Among all of the risk factors cited by the **Landmarks, Framingham** study, and hypertension has been identified as the most potent antecedent of cardiovascular disease. No long term outcome data are available relating blood pressure in childhood or adolescence to cardiovascular risk in adulthood.

A direct relationship exists between blood pressure and left ventricular size in normotensive children and cardiac size increases with

increasing percentiles of blood pressure, suggesting a continuous rather than threshold effect (Burke G, Arohilla R, Cul Pepperaw et al 1987). Evidence from preliminary studies of children and adolescent has shown cardiac ventricular and hemodynamic changes consistent with an adverse effect of mild hypertension, (Dariets S, Meyer R, Loggie J 1990). In addition, in healthy young adults, left ventricular mass has been shown complete closely with plasma angiotensin 2 levels (Harrap S, Dominiczak A 1996).

The adverse effect hypertension on cardiovascular function is compounded by the presence of obesity. The effect of obesity and hypertension on left ventricular mass was investigated in 267 healthy children aged 12 years. The authors concluded that appropriate normalisation of left ventricular mass in necessary for each study population and that left ventricular hypertrophy caused by obesity begins in childhood. In the PADY (Pathobiological Determinants of Atherosclerosis in youth) study after adjustment for other variables, cardiovascular disease risk factor were positively associated with a change in extent of athrosclerosis in the aorta (Mc Gill H, Strong J, Tracy R, et al 1995) Berenson et al like wise reported that specific antemortem risk factors, such as elevation in body mass index, systolic blood pressure, serum low density lipoprotein cholesterol

concentrations, serum Triglyceride concentrations and cigarette smoking are significantly related to the extent of atherosclerotic lesion in young people and that the extent of the atherosclerotic lesion in coronary vessels increased marked by in young people with multiple risk factors. Their finding confirmed that, even in child hood and early adulthood, traditional risk factor for CAD and valid predictors of future cardiovascular events.

Evaluation of the hypertensive patient most hypertension is asymptomatic even after it becomes persistent. This in a way is unfortunate because without symptoms, hypertension is often detected only after target organ damage has occurred, year after onset of disease.

Of the symptoms suggestive of an endocrine etiology (i.e. weight loss, sweating, thrusting, fevers, palpitations, muscle cramps, weakness or constipation), and a family history of primary hypertension and its complications or of genetic disorders known to be associated with secondary hypertension.

The physical examination should focus on evidence of hypertensive encephalopathy , bell's palsy, hypertensive retinopathy , neurofibromas, Ca fe au-lait' spots , lesion in tuberous sclerosis, hirsutism, moon facies, buffalo lump , truncal obesity , striae, the rash of

systemic lupus erythematosus or Henoch – Schonlein purpura, evidence of broncho pulmonary dysplasia, pulmonary edema, congestive heart failure, enlarged kidneys, abdominal mass and edema.

The evaluation includes etiologic investigation and target organ analysis. The target organ aim of the assessment should include fundoscopy and echocardiography, which assist in determining the chronicity and the severity of the hypertension.

Renovascular disease is one of the most common and important causes of childhood hypertension and is potentially amenable to treatment or cure. Approximately 5% to 25% of children with secondary hypertension are said to have renovascular disease (Ingelfinger J 1993). These patients often are found to have high circulating or selective renal vein levels. Historically it has been said that the youngest children with the most severe elevations in systolic blood pressure are more likely to have renovascular disease. Fibromuscular dysplasia is the most common cause of renovascular hypertension in childhood but this varies geographically (Deal J, Snell M, Barratt et al 1992).

In young adults symptoms that are reported in literature headache is the most common, but those who complain of headache are more likely to have their B.P. taken and hypertension discovered. Stewar (1953) found only 17% of patients unaware of their hypertension

complained of headache but among patients with similar level of B.P.'s who were aware of their diagnosis, 71% had headache. This finding is in keeping with the belief that many symptoms described by hypertensives are secondary to anxiety over having the 'silent-killer' as hypertension is frequently described. Anxiety is often expressed as recurrent acute hypertension and chest discomfort. These are the finding of **Degoire et al (1992)**. In another study **copper et al (1985)** found that most symptoms headache in particular, are related not to level of B.P. but rather to anxiety over the diagnosis of hypertension. They found the prevalence of headache among newly diagnosed hypertensives varies little in relation to the levels of B.P. with 15% to 25% having headache whether their diastolic blood pressure were as low as 95 mmHg or as high as 125 mmHg. In another study by **Weiss et al (1972)** neither headache, nor epistaxis 4 tinnitus, dizziness or fainting were more common previously unrecognized hypertension than among those with normal blood pressure.

In a study by **Schooten et al (1986)** retinopathy was found to be an independent indicator of mortality and therefore should be determined in every patient as part of initial examinations and yearly thereafter **Keith, Wagner and Barker (1939)** classified the fundoscopic

changes. Two separate but – related vascular disease are demonstrable in hypertension.

First is hypertensive neuroretinopathy giving rise to haemorrhages, exudates and papilloedema . Second type is arteriosclerotic retinopathy, consisting of arteriosclerotic retinopathy. Consisting of arteriolar narrowing arteriovenous kinking and silver wiring. The original **Keith – Wagner, Barker** grouping mixed the two. In another study of the retinopathy of diabetes punctate and hard exudates is seen in twice as many hypertensive as non hypertensive diabetics. Spontaneous subconjunctival haemorrhages may be a sign of hypertension.

In study of **Bonsa and Thelle (1991)**. Hypertriglyceridemia and hypercholesterolemia are found twice more frequently in untreated hypertensives as in normotensives. The prevalence increases with the level of blood pressure.

Cardiac involvement is signified by a forceful and sustained apical impulse with a forth heart sound (**Frohlich et al , 1992**). Left atrial enlargement may be seen on ECG or echocardiography(**Miller et al , 1988**) LVH as detected by ECG which is much less sensitive and less specific than echocardiography (**Lee et al , 1992**) nonetheless ECG is useful in demonstrating rythm and conduction disturbances , as well as ischemia (**Prisant and Carr, 1993**).

The earliest symptom of renal involvement nocturia and the most commonly identifiable markers of renal involvement are hyperuricemia and microalbuminuria (**Harvey et al 1992**). This may progress to the nephrotic range. Later serum creatinine begins to rise (**Pereger et al 1993**). But the loss of renal function and rise of serum creatinine is asymptomatic thus little absolute increase in serum creatinine will occur until more than 50% of renal function is lost (**Perneger et al 1992**).

Hypertension with diabetes mellitus and hypertension co-exists more commonly than predicted by chance, perhaps 3 times commonly. Of 10% of diabetics with insulin depended form(type I) hypertension is seen in most of the 40% who develop nephropathy , but is seen no more frequently in those who escape nephropathy than in the nondiabetic population (**Norgarol et al ,1990**). In the 90% of diabetics with the insulin independent form (type II) almost all of whom are obese , hypertension is more common than among obese people without diabetes. The connection between hypertension, diabetes and obesity is even stronger in those whose obesity is predominantly in the upper body.

Treatment : The goal of treatment of hypertension in children is to achieve a reduction of blood pressure to below the 95th percentile and prevention of the long term effects of persistent hypertension. The non

pharmacological method means the prevention of obesity, decreasing excessive sodium intake, and exercise. Improvement in physical fitness correlates inversely with blood pressure in young children (**Group B, Basch C, Shea et al 1990**) . Obese adolescents have been shown to have significant reduction in blood pressure with weight loss, and the effect on blood pressure is enhanced when exercise is incorporated into weight – loss program (**Rochini A , Akey J Bobdie D et al 1988**).

The majority of children with secondary hypertension require pharmacological intervention and frequently more than one medication, therapy should be directed at the cause of hypertension, if known children and adolescents who benefit from salt restriction may do well with diet and thiazidediuretic. Patient with high- renin hypertension as well as chronic renal disease and renovascular disease often benefit from ACE inhibitor, calcium channel blocker, B blockers or diuretic can results in reaching the treatment goal of reducing the blood pressure without significant or intolerable side effect (**Ingelfinger J Dillon M 1974**).

AIMS

&

OBJECTIVE

AIMS AND OBJECTIVE

Screening and prevalence of hypertension in school age children and young adults and treatment in Bundelkhand area.

MATERIAL

&

METHODS

MATERIAL AND METHODS

Material: - The study was conducted on the patients who was coming to OPD's of department of medicine and pediatrics, M.L.B. Medical College, Jhansi. The study was also conducted on patients who was admitted in medicine and pediatrics wards and on those patients who was attending the hypertensive clinic we also studied the hypertension through going to different school in Bundelkhand area.

The unit of study was patients attending the hypertensive clinic who had at least 3 or more visits during the period from July 2003 to July 2004. We studied the hypertension in different age groups. We divided these age group into three category.

Category I	6-9 years
Category II	10-12 years
Category III	13-30 Years

Category	Significant hypertension		Severe hypertension	
	Systolic BP (mmHg)	Diastolic BP (mmHg)	Systolic BP(mmHg)	Diastolic BP (mmHg)
6-9 years	>121	>77	>129	>85
10-12 years	>125	>81	>133	>89

In Young adults

13-30 years	Category stage I - 140-159 / 90-99 mmhg
	Stage II - $\geq 160 / \geq 100$ mmHg

The history was recorded in detail for each patient. It included duration of symptoms, when and how was hypertension first detected, its course, family history, dietary history, treatment history complications.

General as well as systemic examination was done to know the general condition, pulse rate, blood pressure, temperature, pallor, icterus, cyanosis, clubbing, oedema, hydration, lymphadenopathy. Systemic examination was done to find out changes due to and associated with hypertension. This included examination of neck, to palpate and auscultate the carotids and thyroid. Examination of heart for size, rhythm and sounds, lungs for rhonchi and rales were examined. The abdomen was examined for renal masses, bruits over aorta or renal arteries. Examination of extremities for peripheral pulses and oedema and neurological assessment were also done.

Methods: - These selected cases were looked for screening and prevalence of hypertension in school age children and young adults and treatment. We measured the BP through auscultation method. In children we used the stethoscope having pediatric bell or diaphragm.

Posture of the patient: We took BP both in lying and sitting position. The patient sat quietly with the back supported for 5 minutes and the arm supported at the level of the heart.

Circumstances:- These criteria was fulfilled while taking the blood pressure .

- ❖ No caffeine during the hour preceding the reading.
- ❖ No smoking during the 15 minutes preceding the reading.
- ❖ No exogenous adrenergic stimulants (e.g. phenylphrine in nasal decongestants or eye drops for pupillary dilation).
- ❖ A quiet; warm sitting.

Equipment: -

Cuff size: - The use of an appropriate sized blood pressure cuff is necessary to ensure accurate measurement. Pediatric cuff series used. We measured the circumference of the upper arm at the mid point between the acromion and olecranon of right arm and selected cuff whose width is approximately two thirds of the distance between

the shoulder and elbow. Use of an appropriately small cuff may falsely elevate the BP reading whereas the use of too large a cuff will give a falsely low reading. However if two cuff's are close in size to measured width of the arm, the large cuff should be selected. Manometer; Mercury manometer is used.

Technique:

Number of Reading: On each occasion we took at least two reading reported by as much times as practical. Where reading varied by more than 5 mmHg, we took additional reading until two are close.

For diagnosis we obtained at least 3sets of reading at least a week apart. Initially we took BP in both arms where it differed we used arm with higher BP.

Performance: We inflated bladder quickly to a pressure 20 mmHg above the systolic pressure, as recognised by disappearance of the radial pulse.

Then we deflated the bladder 3 mmHg every second, and recorded the Korotkoff phase V (disappearance) except in children, in whom we used of phase IV (Muffling). All patients were investigated for: -

Hb	Blood Sugar
TLC	Total Serum Cholesterol
DLC	Serum TG
ESR	HDL

Urine R M	LDL
Blood Urea	VLDL
Serum Creatinine	LDL/HDL ratio
Serum Na +	ECG
Serum K +	Echocardiography USG whole abdomen

WORKING PROFORMA

1. PERSONAL HISTORY

4. Education

5. Address: Present Permanent

6. Occupation

8. Alcoholic / non alcoholic

9. Socio- economic status

10. Sector : Rural / Urban

11. Marital status : Married / unmarried

II. Family History of hypertension/ Diabetes

1. Parental side Age at which detected treatment taken

a. Grand father/ mother

b. Mother/ father

c. Brother/ sister/ cousins

d. Son/ daughter

2. Maternal side :

- a. Grand father/ mother
- b. Mother/ father
- c. Brother/ sister/ cousins
- d. Son/ daughter

3. PRESENT HISTORY**1. Detection of hypertension**

Age at which detected

When

How Course

2. Risk factors

- a. Smoking
- b. Tobacco chewing
- c. Obesity
- d. Alcoholism
- e. Emotional stress
- f. High fat diet
- g. Operation (renal disease)
- h. Diabetes mellitus

3. History of drug intake

- a. Steroids
- b. Oral contraceptives
- c. Vaso pressors
- d. Other(nasal drops, cough mixtures)

4. Treatment taken or not (If Yes , kind of treatment)

Dietary precautions

Drug doses and duration

5. Whether continued / interrupted (if interrupted)

Reasons

ii

IV Routine Diet

- i. Breakfast
- ii. Lunch
- iii. Dinner
- iv. Any added meal
- v. How many times you have used this meal in weeks what type, eggs, meat, butter, milk, sweet

6. Approximate fat consumption in week

Family

Individual

7. Approximate calories / day

1. Face
2. Weight a. Observed b. Ideal
3. Height
4. Pulse a. Radial b. Arterial wall c. Other peripheral pulses
5. BP (systolic/ diastolic)
 - a. Upper limbs Lying Sitting / standing
 - 1st reading
 - 2nd reading

b. Lower Limbs

1st reading

2nd reading

6. Pallor

7. Oedema – Dependent

- Peri orbital

8. Skin Xanthelasma, Tendon Xanthoma

9. Eyes – Arcus senilis

10. Others

SYSTEMIC EXAMINATION

SYMPTOMS

CARDIOSCULAR SYSTEM

Symptoms	Duration	Treatment
Palpitation		
Chest pain		
Dyspnoea / PND		
Cough		
Fainting/ Syncope		

Signs : Mitral area, pulmonary area aortic area

S1

S2

Gallop

S3/ S4

Murmur if any:

Complications :

- a. Left Ventricular failure
- b. Congestive cardiac failure
- c. Ischaemic heart disease
- d. Thromboembolism

LABORATORY INVESTIGATIONS

x- Ray Chest PA view

ECG

ECHO

LAE

PAH

RVH

CENTRAL NERVOUS SYSTEM

Symptoms	Duration	Treatment
Headache		
Vomiting		
Giddiness		
Syncope		

Signs and complications, if any

Stroke (EVA)

Hypertensive encephalopathy

Seizures

TIA(Transient Ischaemic Attack)

Disturbance of speech

Ophthalmic Examination

Blurring of vision / Diminution of vision

Photophobia

Acuity of vision

Fundus – grade of hypertensive retinopathy

Renal:

Kidney palpable / not palpable
Renal artery stenosis , bruit + nt / absent
Evidence of recurrent UTI
Evidence of chronic obstruction
Evidence of acute / chronic renal failure
Renal transplantation
Laboratory Investigations
Urine Routine

M/E

C/S

Blood Urea
Serum Creatinine / Urea creatinine
Serum renin
Serum Ca ++
I.V.P.
Serum Uric acid
Serum Na+, K+
U/S Abdomen
Kidney biopsy

ENDOCRINE AND METABOLIC

a. Diabetes mellitus: IDDM/ NIDDM Yes / No

If yes , duration, Treatment, Blood Sugar : Fasting, PP

b. Evidence of hyperadrenalinism

Cushing syndrome

Pheochromocytoma

c. Evidence of hyper / hypothyroidism

Clinical

Lab

e. Evidence of hypercholesterolemia / hyperglyceridemia

Clinical

Lab investigation

STG

HDL

LDL

VLDL

LDL/HDL ratio

Routine haematological investigations

Hb(gm%)

TLC:- Cells cumm

DLC: P5, L%, E %, M%, B%.

ESR : mm in one hour

OBSERVATIONS

OBSERVATIONS

Table -1

Shown the number of subjects in respective age groups and sex wise distribution respective age groups.

Age groups (years)	Male	Female	Total
6-9	05	01	06
10-12	06	02	08
13-30	28	08	36

Table -2A

Blood pressure age wise distribution in children

		BP value	Age group (6-9 years) (N=6)
Significant Hypertension	Systolic BP	121-128	02
	Diastolic BP	77-84	
Severe Hypertension	Systolic BP	129-132	04
	Diastolic BP	85-89	

This shows 2 subjects having significant hypertension and 4 subjects having severe hypertension.

Table -2B

		BP value (mmHg)	Age group (10- 12years) (N=8)
Significant Hypertension	Systolic BP	125-132	03
	Diastolic BP	81-82	
Severe Hypertension	Systolic BP	>131	05
	Diastolic BP	>89	

This shows subjects having significant hypertension and 5 subjects having severe hypertension.

Table – 3

Age wise distribution of BP according to JNC VII

	Blood Pressure (mmHg)	Age group (13-30 years)
Stage I	140-159	24
	90-99	
Stage II	≥ 160	12
	≥ 100	
Total		36

. Table 3 showing 24 young adults with stage I hypertension and 12 young adults with stage II hypertension.

Table-4

Showing the range of fall systolic blood pressure in children and young adults with antihypertensive treatment

Range of fall in BP (mmHg)	6-9 Years	10-12 Years	13-30 Years
<10	04	03	00
11-19	02	04	10
20-29	00	01	16
30-39	00	00	06
>40	00	00	04
Total	06	08	36

Table shows the distribution of the fall of systolic blood pressure in one month 3 consecutive reading BP with one or more drugs. The maximum 17 subjects had a fall in range of 20-28 mmHg out of 50 subjects.

Table -5

Showing the range of fall of the diastolic pressure with antihypertensive treatment on 3 successive reading in one month on one or more drugs.

Range of fall in BP (mmHg)	6-9 Years	10-12 Years	13-30 Years
0-9	03	05	10
10-19	03	03	17
20-29	00	0	05
>30	00	00	04
Total	06	08	36

Table shows the fall in diastolic blood pressure, the greater number of subjects (23) had a fall between 10-19 mmHg. Only 4 patients required a lowering of greater than 30mmHg to normalize the diastolic pressure.

Table- 6

**Distribution of cases according to antihypertensive drugs given
some patients were on more than one drug (N= 50)**

Drugs	Age group in years		
	6-9	10-12	13-30
Calciumm channel blocker			
Claiss I (Nefidepine)	00	01	02
Amlodepin	01	00	02
β blockers	00	00	24
Atenolol / Metoprolol			
Diuretcs	02	03	04
Aldomet	00	00	00
α – blocker	00	00	00
ACE Inhibitors	03	04	04
Total	06	08	36

The various drugs used have been shown in table. Nefidepine (3 out of 50) and amlodepin (3 out of 50), and Betablockers (24 out of 50) were the drug most commonly used. The total number of

patients on calcium channel blockers were 6, on diuretics 9, on aldomet 0, on α Blockers 0 and on ACE inhibitor 11.

Table -7
Showing the symptoms reported by the patients (N=50)

Sl. No	Symptoms	No. of cases
1	Headache	30
2	Giddiness	09
3	Palpitation	03
4	Chest pain	02
5	Oedema	15

The symptoms which were commonly reported by patients are shown in the table. Headache was present in 30 subjects, 9 patients reported giddiness, palpitation was reported by 3 subjects, 2 patients give the history of chest pain during the course of their hypertension, oedema was reported by 15 patients.

Table -8
Showing the STC level in various age group (mg/dl) (N=50)

STC levels mg/dl	Age group (years)		
	6-9	10-12	13-30
<200	06	08	34
>200	00	00	02

In this table it can be seen that 2 patients had a STC level >200 and 48 subjects out of 50 were in normal level.

Table- 9
Showing STG levels (mg/dl) in various age group (N=50)

Age group (years)			
STG levels (mg/dl)	6-9	10-12	13-30
<150	06	08	33
>150	00	00	03

Table shows the distribution of serum triglycerides in the various age groups 3 subjects have a value higher than 150 mg/dl., 47 subjects have value <150 mg/dl.

Table -10
Shows the VLDL level in various age groups (mg/dl) (N=50)

Age group (years)			
VLDL levels (mg/dl)	6-9	10-12	13-30
<40	06	08	34
>40	00	00	02

Table shows the distribution of VLDL in various age groups, 2 subjects have a higher value > 40 mg/dl.

Table -11
Showing the LDL levels in various age groups (N=50)

LDL levels (mg/dl)	Age group (years)		
	6-9	10-12	13-30
<130	06	08	34
>130	00	00	02

Table showing the distribution of LDL levels in various age groups. 2 subjects having higher value >130 mg/dl. 48 subjects have the value <130 mg/dl out of total 50.

Table -12
Showing distribution of HDL level in various age group.

HDL levels (mg/dl)	Age group (years)		
	6-9	10-12	13-30
25-29	00	00	00
30-34	00	00	00
35-39	02	01	03
40-44	04	05	30
45-49	00	02	03
>50	00	00	00

Table –13
Showing the LDL / HDL ratio in the group of patients (N=50)

LDL/HDL levels	No. of cases
<5	48
>5	02

Table –14
Showing the blood urea level (mg/dl) in various age groups
(N=50)

Blood urea (Mg/dl)	Age group (years)		
	6-9	10-12	13-30
<25	01	011	21
25-29	00	01	05
30-34	00	00	02
35-39	00	00	02
>40	05	06	06

Table showing distribution of blood urea in the different age groups.
17 subjects have a higher value (> 40 mg/dl). The range 15-40 was taken as normal.

Table –15
Showing the serum creatinine level (mg/dl) in various age groups (N=50)

Age group (years)				
		6-9	10-12	13-30
Serum creatinine (Mg/dl)				
<0.5	01	00	14	
0.5-0.9	00	01	16	
1 –1.5	00	01	02	
> 1.5	05	06	04	

Table showing the distribution of serum creatinine levels in the various age group. 15 subjects having higher value (>1.5 mg/dl) out of total 50. So 15 subjects having the renal failure. All other values were in the range of 0.5 to 1.5 mg/dl.

Table –16
Showing the Hb levels (gm%) in various age groups (N=50)

Age group (years)				
		6-9	10-12	13-30
Hb gm%				
5-7.9	02	00	00	
8-10.9	00	02	02	
11-13	01	01	04	
>13	03	05	30	

Table shows the distribution of Hb level in various age groups. A total of 6 subjects had level below 11gm%. Out of these 6 subjects, 2 were in the severely anemic range of 5 to 7.9 gm%.

Table –17
Showing the finding of urinary examination of hypertensive patients

Age group (years)			
Urinary Finding	6-9	10-12	13-30
Albumin (+) or more	05	06	04

Table shows the urinary finding observed in the study group . 15 patients tested positive for albumin.

Table – 18
Showing the ECG, ECHO and USG abdomen finding in various age groups.

	Normal	Abnormal
ECG	47	03
ECHO	48	02
USG abdomen B/L renal parenchymal disease	35	15

Table shows the 3 subjects having abnormal ECG finding, 2 subjects having abnormal ECHO, On USG abdomen 15 subjects having B/L renal parenchymal disease.

Table –19
Showing the distribution of patients according to their BMI
(N=50)

	Number of cases
1. Patients with body weight lower than ideal range for their height BMI <20.	40
2. Normal range 20-25	06
3. more than normal range	04

Table shows the various group based on body mass index (weight in kg/ height²) distribution. 4 patients out of total 50 subjects had a BMI > 25 which is taken as abnormal. 6 were in the normal range having a BMI 20-25.

Table –20
Showing the complications observed in various age groups
(N=50)

complications	Age group (years)		
	6-9	10-12	13-30
Renal failure	05	06	04
CVA	00	00	00
MI	00	00	00
IHD	00	00	00
Retinopathy	00	00	00

Table showing the different complications observed. The commonest was renal failure with a total number of 15 subjects out of 50 being affected.

DISCUSSION

DISCUSSION

This study had the majority of the patients with increasing incidence of hypertension with age. **Corneni – Huntly et al** noted 5% increase for each 10 years interval of age from the base line . The number of male subjects was slightly higher in this study. In our study number of male children was 11, whereas females children was 3 in number. In young adults the male and female was 28Vs 8. In **NHANES-II** survey in the United States, the percentage of male was always higher at all ages. Hence it is evident that the age and sexwise distribution seen in this group of 50 patients was similar to those observed in other studies.

In this study we screened total 800 school age children in which we found 14 children hypertensive, so the prevalence rate in this group has been found to be 1.7% .

In Young adults we screened total 1400 young adults in which we found 36 young adults hypertensive , so the prevalence rate was found to be 2.5%. **Sinaiko et al** found the prevalence of hypertension to be 2% among 14000 school age children. In a cohort of Junior High School Students the prevalence was found to be 4.2% at initial examination to 1% on repeated examinations. **NHANES** in the united

states prevalence rate was found 4% in the age group 18 to 24 years.

In school age children the most common cause of hypertension is secondary hypertension and out of the causes of secondary hypertension most common is renal disease. In our we study we found secondary hypertension in 78.5% of school age children (11Vs14) . Out of these children 72% cases had renal parenchymal disease (9Vs 11). And 18% was having renovascular disease (2Vs11). **Ingelfinger** 1994 evaluated the young patients and found that approximately 60 to 80% of secondary hypertension in children was caused by the renal parenchymal disease and renovascular disease had been found in approximately 5 to 25% children.

In young adults essential hypertension was found in 88.8% cases (32Vs 36) . **Sinaiko et al** 1989 found essential hypertension in 90 % of young adults. So it is evident that essential hypertension seen in most of the young adults. In our study is similar to those observed in other studies.

In school age children severe hypertension was found 9 subjects (64.2%) and significant hypertension was found in 5 subjects (35.7%). In young adults stage I hypertension was found in 24

subjects (66.6%) and stage II hypertension was found in 10 subjects (33.3%) .. Other workers in the hypertension detection and follow up programme noted the highest incidence of stage I hypertension (67%). When we consider blood urea levels, 11 children had elevated blood urea level ($>40\text{mg/dl}$) and 6 young adults having higher value ($>40\text{mg/dl}$) . when we consider serum creatinine levels, 11 children had elevated serum creatinine level($>1.5\text{mg/dl}$) and 4 young adults had elevated serum creatinine level ($>1.5\text{mg/dl}$). When we consider the urinary albumin levels, 11 children had tested positive for urinary albumin, and in young adults, 4 subjects was tested positive for urinary albumin. This shows that secondary hypertension in childhood is mainly caused by the renal parenchymal disease. **Ingelfinger et al** shows that approximately 60to80% of secondary hypertension in childhood is caused by renal parenchymal disease.

When we consider the lipid profile 2 subjects had elevated serum total cholesterol levels ($>200\text{mg/dl}$) and 3 subjects had elevated serumtriglyceride levels ($>150\text{mg/dl}$) and 2 subjects had elevated low density lipoprotein levels so these data indicates increase in incidence of hypercholesterolemia in hypertensive patients. Several well-conducted epidemiological studies have

demonstrated that cholesterol levels was found to be significantly higher in hypertensive patients.

Headache was a common symptom reported by the subjects (60%). The value lies between the 17% incidence of headache in previously undiagnosed hypertentives and 71% incidence of headache in diagnosed (**Stuart, 1953**).

Oedema which is a definitive physical sign with no functional correlation was present in 15 subjects out of 50 patients. All from amongst those who went into renal failure.

Obesity is also related to hypertension 4 out of 50 subjects had BMI > 25, which is taken as abnormal. The association of obesity and hypertension is believed to be causal one, where in obesity contributes not only to higher blood pressure in children and adolescents but also has been shown to have a significant predictive value for its association with other cardiovascular risk factors such as cholesterol levels and serum lipoprotein ratio. Between 13% and 36% of 12 to 17 years old adolescent was obese , depending on gender and race, and additional 4% to 12%, may be considered superobese (**Fugurora , Colon R 1997**).

Leftventricular diastolic dysfunction is also related to the age of the patients, as this increases with advancing age irrespective of the duration of hypertension, by systolic dysfunction has no correlation (**Savage et al 1990**). The most common effect of hypertension on heart is hypertrophy of left ventricle. Incidence of LVH increases with advancing age in hypertensive cases. In this study only 2 young adults had LVH. Because our study was done in school age children and young adults so incidence of LVH was found lower in these age groups.

The international society of hypertension proposes that any of the five category of drugs should be suitable for an initial therapy. Obesity prevented by decreasing excessive sodium intake, and exercise. Improvement in physical fitness correlates inversely with blood pressure in young children. Obese adolescent have been shown to have significant reduction in blood pressure with weight loss, and effect on blood pressure is chanced when exercise is incorporated into weight loss programme (**Rocchin A, Anderson et al 1988**). In young adults role of β - bloker was effective to bring down the blood pressure in a normal range

In 48% cases the role of β blockers found effective. In school age children the role of diuretics and ACE inhibitor was effective. In 22% cases ACE inhibitors and 18% cases diuretic was effective to bring down the blood pressure in a normal range. Other drugs were selected depending on the patients profile. About 50% of patients required a reduction of 20-30 mmHg systolic and 10-19 mmHg diastolic blood pressure. This was achieved in most patients. this was in accordance with the current recommendation of WHO and International Society of hypertension.

CONCLUSION

CONCLUSION

1. The conclusions of the current study were: Blood pressure is slightly higher in boy's then in girls during the first decade of life. This difference begins to widens around the onset of puberty, and blood pressure significantly higher in young men by the end of teenage years. School age children (11Vs 3) and in young adults (28Vs8).
2. Prevalence of hypertension increases with age. In this study the prevalence rate in school age children was 1.7%. We found the 14 children having hypertension, whereas in young adult prevalence rate was 2.5%. We found 36 young adults having hypertension.
3. Secondary hypertension more commonly found in school age children and caused by renal parenchymal and renovascular disease in most of cases. In this study we found 11 subjects with secondary hypertension (78.5%), 9 subjects was having renal parenchymal disease (72%) and two was the cases of renovascular disease (18%).
4. Essential hypertension is most commonly found in young adults. It was found in 88.8% cases. Total 32 cases out of 36 have essential hypertension.

5. Obesity contributes to essential hypertension in young adults.

Total 4 subjects out of 36 having their BMI >25 . Obesity also related to hypercholesterolemia and hypertriglyceridemia, 2 subjects was having STC > 200 mg/l and 3 subjects was having STG > 150 mg/dl.

6. The symptoms commonly observed in this group of patients were headache (60%), giddiness 18%, palpitation (6%), chest pain (4%) and oedema (30%).

7. In young adults role of β blockers as effective in 48% cases. In children the role of diuretics and ACE inhibitors was effective. In 22% case ACE inhibitors and in 18% case diuretics was effective.

8. Most common complication of hypertension below age 30 year was renal failure. 15 subjects (30%) of renal failure shows a value of serum creatinine higher than 1.5 mg/dl.

BIBLIOGRAPHY

BIBLIOGRAPHY

1. Blood pressure Measurement in children: From the department of pediatrics Nephrology, University of Wisconsin children's hospital, Madison, Wisconsin (SMB) and the division of Pediatric Nephrology University of chicago children hospital, chicago I Illinois (AJA).
2. National Heart, Lung and blood Institute: Uptake on the 1987. Task force on high blood pressure in children 1987, pediatrics 79:1,1997.
3. Bergulaud G, Anderson O, Wilhmsen L : Prevalence of primary and secondary hypertension. BMJ 1976,2:554-556.
4. Burt VL , Culter JA, Higginus M et al : Trends in the prevalence awareness , treatment and control of hypertension the adult US population . Hypertension 1995 a, 26:60-69.
5. National High Blood Pressure education program working group. Areh Intern. Med 1993; 153:186-208.
6. Epidemiology of hypertension from childhood to young adulthood in black, white, and Hispanic population samples. Public Health Rep 113:3,1996.
7. Fredich ED, Grme , Lab arthe DR, Maxwell MH, Perloff D, and Weidman WH . Recommendations for human blood pressure association. Hypertension 1988; 11:209A-222A.
8. Petric JC, O ' Brien ET, Little WA and De Swiet M. Recommendations on blood pressure measurement. Br. Med. J. 1986,293; 611-615.

9. Hirsh J-M, H edner J, Wernsted L, LundbergJ, and Hedner T, Hemodynamic effects of the use of oral snuff. Clin pharmacol Ther, 1992; 52:394-401.
10. Shi J, Benowitz NL, Denaroel and Schiner LB, pharmacokinetic – pharmacodynamic modelling of caffeine: Tolerance to pressor effects, Clin, pharmacol. Ther 1993; 53-6-14.
11. Potter JF, Watson RDS, Skanw and Beevers DG: The pressure and metabolic effect of alcohol in normotensive subjects. Hypertension 1986;8;625-631.
12. Gillman MW, Ellison RC: Childhood prevention of essential hypertension. Pediatr Clin North Arm 40:179,1993.
13. Figtteroa. Colon R, Franklin F, Lee J et al : Prevalence of obesity with increases blood pressure in elementary school age children , South Med J 90:806,1997.
14. Benetos A, Thomas F, safer ME, et al: Should diastolic and systolic blood pressure be considered for cardiovascular risk evaluation. J Am Coll Cardiol 2001, 37:163-168.
15. Rosner B, Prineas R, Loggie J et al, blood Pressure horniograms for children and adolescents by height, sex and age in United States; J Pediatr 123:871,1993.
16. Burke G, Arcilla R, Culpepper W, et al: Blood Pressure and echocardiographic measures children: The Bogalusa Heart Study. Circulation 75:106,1987.
17. Daniels S, Meyer R, Loggie J: Determinants of cardiac involvement in children and adolescent with essential hypertension. Circulation 82:1243,1990.

18. Deal J, Snell M, Barratt T et al: Renovascular disease in childhood. *J Pediatr* 121:378, 1992.
19. Ingelfinger J, Dillon M: Evaluation of secondary hypertension. In Holiday - Barrat A (ed) *Peditric Nephrology*, ed3, Baltimore, Williams and Wikkins, 1994, P1146.
20. Ingel Finger J: Nephrology from - Renovascular hypertension in children. *Kidney Int* 43; 493, 1993.
21. Labiathe D, Muller W, Eissa M: Blood Pressure and obesity in childhood and adolescence Epidemiologic aspects . *Ann Epidemiol* : 337, 1991.
22. Libermen E : Pediatric hypertension: Clinical perspective *Mayo Clin Proc* 69:1098, 1994.
23. Mc Gill H, Strong J, Tracy R, et al: Pathological determinants of atherosclerosis in Youth (PDAY) Research Group: Relation of Postmortem renal index of hypertension to atherosclerosis in youth *arterioscler Thromb Vasc. Biol* 15:2222, 1995.
24. National Heart, Lung and Blood institute:Update on the 1987 Task Force on High Blood Pressure in children and Adolescents: A working group from the national High Blood Pressure education Program. *Pediatrics* 9.8:649, 1996.
25. Rabin Owitz A, Kushner H, Faulkner B: Racial differences in blood pressure among Urban adolescents . *J Adolesc Health* 14:314, 1993.
26. Berenson G, Wattigney W, Tracy R. et al. Atherosclerosis of the aorta and coronary arteries and cardiovascular risk factors in persons aged 6 to 30 years and studied at necropsy (the Bogalusa Heart study). *Am J Cardiol* 70'851, 1992.

27. Burns T, Moll P, Lauer R: Increased Familial cardiovascular mortality in obese school children. The Muscatine Ponderosity Family study Pediatrics 89,262,1992.
28. Geleijnse J, Grobbee D, Hofman A: Sodium and potassium intake and blood pressure changes in childhood , BMJ 300:899,1990.
29. Lenfant C, Savage P. The Early natural history of atherosclerosis and hypertension in the young: National Institutes of health Perspectives. Am J Med Sci 310(Suppl 1) 3,1995.
30. Mahoney L, Clarke W, Burns T, et al: childhood predictors of high blood pressure Am J Hypertens 4:6085,1991.
31. Mongeau JG; Pathogenesis of essential hypertension. Pediatr Nephrol 5:404, 1991.
32. Rocchini A, Katch V, Anderson., et al: Blood pressure and obese adolescents: Effect of weight loss Pediatrics 82:116,1988.
33. Rocchini A, Kev J, Bondie D, et al: The effects of weight loss on the sensitivity of blood pressure to sodium in obese adolescents. N. Engl J Med 321:580,1989.
34. Rosner B, Prineas R, Loggie J, et al: Blood pressure nomogram for children and adolescent by height, sex, and age in the United States, J Pediatr 123; 871,1993.
35. Schieken R, Genetic factors that predispose the child to develop hypertension Pediatr Clin North Am 40:1,1993.
36. Shear C, Burke G, Freedman D et al: Value of childhood blood pressure measurements and family history in predicting future blood pressure status: Results from 8 years of follow-up in the Bogalusa Heart Study.Pediatrics. 77:862,1986.

37. Sinaiko A: Hypertension in children N Engl J Med 335:1968, 1996.
38. Sinako A, Gomez- Marin O, Prineas R, Effect of low sodium diet or potassium supplementation on adolescent blood pressure. Hypertension 21;1989,1993.
39. Sinako A, Gomez- Marin O, Prineas R: Prevalence of "significant" hypertension in junior high school -aged children. The Children and adolescent Blood Pressure Program. J. Pediatr 114:664,1989.
40. Voors A, Bereson G, Dalferes E Jr, et al: Racial differences in Blood pressure control. Science 204:1091,1979.
41. William D, Going S, Lohman T. et al: Body fatness and risk for elevated blood pressure, total cholesterol, and serum lipoprotein ratios in children and adolescents. Am J Public Health 82:358,1992.